Effects of Spinosad, Spinosad Bait, and Chloronicotinyl Insecticides on Mortality and Control of Adult and Larval Western Cherry Fruit Fly (Diptera: Tephritidae)

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J. Econ. Entomol. 99(5): 1722–1732 (2006)

ABSTRACT Effects of spinosad, spinosad bait, and the chloronicotinyl insecticides imidacloprid and thiacloprid on mortality of the adults and larvae of western cherry fruit fly, *Rhagoletis indifferens* Curran (Diptera: Tephritidae), were determined in the laboratory and the field. Spinosad and spinosad bait caused higher adult mortality than imidacloprid, which caused higher mortality than thiacloprid. Only spinosad bait prevented oviposition. All materials were more toxic to adults when ingested than when topically applied. Spinosad bait had the greatest residual toxicity on leaves, killing 100% of adults when aged for 14 d in the field. When materials were sprayed on infested cherries, numbers of live larvae in fruit after 8 d were lower in imidacloprid and thiacloprid than in spinosad and spinosad bait treatments, which did not differ from the control, but all materials reduced larval emergence over 30 d. In the field, spinosad and spinosad bait were as effective in suppressing larval infestations as azinphos-methyl and carbaryl, whereas imidacloprid was effective in most cases and thiacloprid was generally less effective than azinphos-methyl and carbaryl. Overall, results in the laboratory and field show that spinosad and chloronicotinyl insecticides differed significantly in their effectiveness against adults and larvae of *R. indifferens* but that spinosad, spinosad bait, and imidacloprid seem to be acceptable substitutes for organophosphate and carbamate insecticides for controlling this fruit fly.

KEY WORDS *Rhagoletis indifferens*, spinosad, imidacloprid, thiacloprid, control

Insecticides continue to be vital in efforts to control the western cherry fruit fly, *Rhagoletis indifferens* Curran (Diptera: Tephritidae), the most serious insect pest of commercial sweet and sour cherries, *Prunus avium* (L.) L., and *Prunus cerasus* L., respectively, in the western United States. Organophosphate and carbamate insecticides have been used for many years to successfully control this fly in Washington (Eide et al. 1949, Johansen et al. 1954, Frick et al. 1954, Frick 1957), Oregon (Zwick et al. 1970, 1975), Utah and other western states, and in British Columbia (Raine and Andison 1958). The zero tolerance for fly larvae in cherries (Anonymous 1968) has necessitated the use of these highly toxic insecticides in commercial orchards. Isolated homeowner or abandoned trees can be heavily infested and also need to be treated with these insecticides to reduce chances of flies dispersing to commercial orchards. However, because of their impending loss or restricted use in the future due to the Food Quality and Protection Act (1996), newer insecticides are increasingly important in efforts to control the fly. These insecticides include various formulations of a macrocyclic lactone, spinosad, which is derived from the soil bacterium *Saccharopolyspora spinosa* Mertz and Yao; and two chloronicotinyl insecticides, imidacloprid and thiacloprid (Hu et al. 1998, 2000; Liburd et al. 2003; Reissig 2003; Barry and Polavarapu 2005). Spinosad is also used as the toxin in bait sprays (Reissig 2003, Yee and Chapman 2005). These insecticides have been tested against adult apple maggot, *Rhagoletis pomonella* (Walsh) (Reissig 2003), and adult blueberry maggot, *R. mendax* Curran (Liburd et al. 2003, Barry and Polavarapu 2005, Barry et al. 2005). In *R. pomonella,* spinosad was effective in the laboratory, although it performed inconsistently in the field, whereas spinosad bait was ineffective in the field. Imidacloprid was effective in the laboratory and not effective in the field. Thiacloprid was ineffective in the laboratory, but it was effective in the field (Reissig 2003). In *R. mendax,* spinosad, imidacloprid, or thiacloprid were equally effective (Liburd et al. 2003, Barry and Polavarapu 2005, Barry et al. 2005). In the walnut husk fly, *R. completa* Cresson, spinosad bait was also effective, but it did not eliminate infestations (Van Steenwyk et al. 2003), which was also true in *R. indifferens* (Yee and Chapman 2005).

In addition to killing the adult fly and therefore reducing oviposition, imidacloprid and thiacloprid may kill larvae inside fruit to some extent because both materials are absorbed by plant tissue (Elbert et al., 1991, 2001). Controlling eggs and larvae is important

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because infestations in unpicked cherries after harvest frequently approach 100% (Frick and Simkover 1953). Organophosphates such as parathion (Frick and Simkover 1953) and dimethoate (Zwick et al. 1975) are known to kill larval *R. indifferens* inside fruit, but spinosad and the chloronicotinyl insecticides have not been evaluated for their effects on larvae of this species, although larvae of *R. completa* were controlled to some extent by imidacloprid (Van Steenwyk and Coates 1998). Determining the effects of these insecticides on larvae will result in a more complete understanding of how the materials work in reducing *R. indifferens* populations.

The objectives of this study were to determine the effects of spinosad, spinosad bait, imidacloprid, and thiacloprid on mortality of adult and immature *R. indifferens* and their effects on factors related to mortality in the laboratory. Adult mortality, topical and ingestion modes of adult kill, oviposition, residual toxicity, and larval emergence after insecticide treatments were determined. Results of field efficacy tests conducted in 2000–2005 also are reported for comparison with laboratory findings.

Materials and Methods

Fly Source and Experimental Conditions. Six experiments were conducted: four in the laboratory with adults (experiments 1–4); one in the laboratory and field with larvae (experiment 5); and one in the field, which consisted of a series of spray trials (experiment 6). In experiments 1–3, flies originated as larvae collected from cherries in central Washington in June and July 2003 and 2004. Puparia were chilled at 3°C for 4–8 mo. They were then transferred to 27°C for adult emergence. Adults were maintained after emergence on a dry 80% sucrose/20% yeast extract (wt:wt) diet (EZ Mix, Sigma-Aldrich, St. Louis, MO) on paper strips (referred to as "food" hereafter) and water on wicks inside 473-ml paper containers. In experiment 4, flies were collected as adults from the field (same areas as experiments 1–3). In experiment 5, naturally infested cherries were collected. Unless noted, experiments 1–4 were conducted at 25–27°C, 30–40% RH, and a photoperiod of 16:8 (L:D) h.

Materials Tested in Laboratory Studies. Spinosad (Entrust, 80.0% spinosad, Dow AgroSciences LLC, Indianapolis, IN) and/or spinosad bait (GF-120 NF Naturalyte Fruit Fly Bait, 0.02% spinosad, Dow AgroSciences LLC), imidacloprid (Provado 1.6 Flowable, 17.4% imidacloprid, Bayer CropScience, Research Triangle Park, NC), and thiacloprid (Calypso 4 Flowable, 40.4% thiacloprid, Bayer CropScience, Research Triangle Park, NC) were evaluated in all laboratory experiments. Each material was tested using the highest label rate, by using weight:volume rather than volume: volume due to the small insecticide quantities needed. For spinosad, the rate was equivalent to 17.0 g/378 liters (32 ppm AI); spinosad bait, 88.7 ml (106.4 g)/532 ml (89 ppm AI); imidacloprid, 56.7 g/378 liters (26 ppm AI); and thiacloprid, 56.7 g/378 liters (61 ppm AI). All materials were weighed on a balance (AB104, Mettler, Toledo, Switzerland). Insecticides were mixed immediately in deionized water and tested within 3 h.

Experiment 1: Effects of Insecticides and Bait on Adult Mortality. Ten male and 10 female flies 2–7 d old were placed inside a 473-ml paper container with food and water with no insecticides (control) or with 100 μl (test 1A) or 500 μl (test 1B) of each of the four treatments. Treatments were presented as five (test 1A) or 25 (test 1B) 20-μl drops spread uniformly on a shallow plastic dish (5.0 cm in diameter) on the bottom of a container over 4 d. Fly mortality was determined at 1, 2, 3, and 4 d after exposure. Flies were classified as dead if they could not walk (the same criterion was used for all adult experiments). There were three or four replicates of the control and treatments in test 1A and five of each in test 1B.

Experiment 2: Effects of Insecticide- and Bait-Treated Cherries on Adult Mortality and Oviposition. Twenty (10 females, 10 males) (test 2A) or 10 (five females, five males) (test 2B) 13–27-d-old flies were placed inside a 473-ml paper container with food, water, and three cherries that had been dipped in water (control) or solutions of spinosad, spinosad bait, imidacloprid, or thiacloprid for 2–3 s. Cherries were placed on a shallow plastic dish (5 cm in diameter). The spinosad bait was allowed to drip for a few seconds from the cherries before being placed on a dish. In test 2A, cherries were exposed to flies for 4 d. Mortality was recorded at 1, 2, 3, and 4 d after first exposure. There were six replicates of the control and treatments. All cherries were stored in alcohol for later dissections to determine egg numbers. In test 2B, cherries were exposed to flies for 2 d. Mortality was recorded at 1, 2, and 6 d after first exposure. Cherries from three of six replicates were dissected to determine numbers of eggs laid over the 2 d. Cherries from the other three replicates were set aside in 473-ml plastic containers to determine larval emergence by counting puparia after 4 wk.

Experiment 3: Effects of Topical Application and Ingestion of Insecticides on Adult Mortality. Spinosad, imidacloprid, and thiacloprid were mixed with 20% sucrose (wt:wt) to stimulate feeding and tested against 3–11-d-old flies. Each treatment was included in every age within this range. The control was 20% sucrose only. Newly emerged flies were held inside 473-ml paper containers with food and water. Food was removed 16–20 h before tests. Individual flies were immobilized at 1.7–3.3°C for 5–6 min. For testing effects of topical application, a 2-μl drop of solution was placed on top of the thorax of a single fly under a microscope. For ingestion effects, a 2-μl drop of solution was placed in a 5.0- by 1.4-cm glass vial 15 min after a fly was introduced into the vial. The fly was closely observed and given a maximum of 15 min to drink the solution. All flies drank the insecticide solutions within this time, some consuming the entire 2-μl drop. After topical application and ingestion treatments, each fly was placed inside a 473-ml paper container with food and water. Mortality was checked daily up to 30 d. There were 15–25 flies of each sex for the control and treatments.

Experiment 4. Effects of Aging Insecticide and Bait Residues on Adult Mortality. Spinosad, spinosad bait, imidacloprid, and thiacloprid aged for 0, 3, 7, and 14 d on sweet cherry leaves in an experimental orchard in Moxee, WA, were exposed to field-collected flies in 2005. A hand-held sprayer was used to deliver ≈10 ml of spinosad bait and 20 ml of the other materials to tops and bottoms of ≈30 leaves on the south sides of trees on 6, 13, 17, and 20 June. Bait sprays are not intended to provide 100% coverage and thus a lower spray volume of spinosad bait was used. Unsprayed leaves served as controls. Mean high and low temperatures ± SE during the 14 d were 23.3 ± 0.8 and 6.6 ± 0.5°C, respectively. There was 1.3 mm of precipitation on 7 June and a trace amount on 18 June. Most days were sunny. For the "0" day treatment, leaves were sprayed and then air-dried for 1 h before being exposed to flies. Flies were collected from Kennewick and Zillah, WA, over a 2.5-wk period before the experiment and maintained at 20–21°C with food and water inside 473-ml paper containers. Each container (replicate) held eight male and four female flies. Flies that died before the start of the experiment were replaced as needed. For each replicate, one randomly chosen control or treated leaf (28–42 cm^2) was placed inside a container. The leaf was laid on its edge to maximize exposure of flies to insecticide residues on both sides of the leaf. Flies were provided with food and water. Mortality was checked at 1, 3, and 7 d after fly exposure to the leaves. There were five replicates of the control and each treatment. The experiment was conducted at 24–29°C.

Experiment 5: Effects of Spraying Cherries with Insecticides and Bait on Larval Mortality and Emergence. Sweet cherries with stems attached were picked from five infested trees on 7 and 8 June 2005 in Kennewick and treated with water (control), spinosad, spinosad bait, imidacloprid, and thiacloprid on 8 June. The cherries were ripe, dark red, and 2–2.5 cm in diameter. There were 110 cherries for the control and each treatment from each tree. The 110 cherries were placed on hardware cloth and then sprayed with 10 ml of each material by using a squirt bottle. They were then suspended above a tub containing dry soil and held outdoors in the shade for 30 d (8 June–8 July). At 8 d, 10 cherries were randomly selected from each sample and opened to determine numbers of dead and live larvae. Each larva was measured to determine whether growth was affected. The other 100 cherries were held on the hardware cloth for an additional 22 d. At 15 and 30 d after treatment, numbers of puparia in the soil at the bottom of each tub were counted. Puparia were stored in sealed cups at 21°C in moist soil. All puparia were dissected at 30–37 d posttreatment (within 1 wk of the end of the experiment) to determine mortality. There were five replicates of the control and treatments, with one replicate being a sample of 110 fruit from each of the five trees.

Experiment 6: Effects of Insecticide and Bait Sprays on Adult and Larval Control in the Field. To compare the efficacy of spinosad, chloronicotinyl, and organophosphate insecticides for control of adult flies and larvae in the field, spray trials were conducted in Utah in 2000–2005 and in Washington in 2004 (Table 1). In Utah, six trials were conducted in 'Montmorency' sour cherry at the Utah State University Horticultural Research Farm in Kaysville (Davis County). Plot and treatment specifications for trials are provided in Table 1.

Insecticides were applied with an orchard air-blast sprayer (654–748 liters/ha), except for the spinosad bait, which was applied at 1.5 liters/ha with a handgun sprayer mounted on an all-terrain vehicle driven at a speed of 11.3 km/h. A control treatment without insecticides was included in all trials. In trials 1 and 2, treatment design was a randomized complete block with four replications. In trials 3–6, treatments were replicated four (trials 4 and 6) or six (trials 3 and 5) times, but they were not randomized. A randomized block design was not used in trials 3–6 to accommodate creating larger plots (0.2–0.9 ha) to avoid interplot interference among insecticide treatment effects observed in trials 1 and 2.

Application of insecticide treatments was initiated within 7 d of the first adult capture on yellow sticky traps (Pherocon AM, Trécé Incorporation, Adair, OK). Ammonium carbonate (7.0 ± 0.6 g in powder form) was enclosed in a round plastic container (38 mm in diameter by 20 mm in height) with a small hole in the lid and attached to a bottom corner of each trap to serve as an additional attractant. One trap was placed at a height of 1.8 m and on the south side of each of two trees in each replicate before emergence of the first adults through early to mid-August. First date of adult catch occurred from 17 to 27 May across the 6 yr. The numbers of adults per trap were determined one to two times per week. Traps were replaced every 2 to 3 weeks when the adhesive was diminished. Ammonium carbonate containers were refilled as needed.

Fruit samples were collected to assess infestation with larvae when fruit were immature (one time in late June in trials 1–3 [400–450 fruit per treatment] or weekly in trials 4–6 [400–600 fruit per treatment per date]) and mature (1,000–2,400 fruit per treatment on 30 June to 17 July across years [composed of 500–600 fruit per subsample from two (trials 1 and 6), three (trials 2, 3, and 5), or four (trial 4) subsamples per treatment]). Immature fruit were placed on larval emergence trays (hardware cloth placed over plastic bins) to collect emerging larvae, and mature fruit were dissected and the numbers of larvae counted.

In Washington, one spray trial (trial 7) was conducted in 2004 in a sweet cherry ('Bing') orchard at the USDA experiment station in Moxee, WA, by using single trees (Table 1), which simulated unmanaged homeowners' trees. Yellow sticky traps baited with ammonium carbonate were placed in selected trees in May to detect first fly emergence. A control and spinosad, spinosad bait, imidacloprid, and thiacloprid treatments were compared. The test was set up as a

Table 1. Plot and treatment specifications for field insecticide and bait efficacy trials conducted in Utah during 2000–2005 (trials 1–6) and in Washington in 2004 (trial 7)

Trial	Yr	Location	Plot size (ha)	Treatment: insecticide common name (formulation, % [AI])	Rate	Reapplication interval (d)	No. applications
1	2000	Kaysville, UT	0.1	Control			
				Thiacloprid (Calypso 4F, 40.4%)[a]	0.3 liter/ha	14	3
				Imidacloprid (Provado 1.6F, 17.4%)[a]	0.6 liter/ha	14	3
				Spinosad (Success, 22.8%)[b]	0.4 liter/ha	14	3
				Spinosad (Success, 22.8%)[b]	0.4 liter/ha	7	5
				Azinphos-methyl (Guthion 50WP, 50%)[c]	1.7 kg/ha	14	2
				and Carbaryl (Sevin XLR Plus, 44.1%)[c]	4.7 liters/ha		1
2	2001	Kaysville, UT	0.1	Control			
				Thiacloprid (Calypso 4F, 40.4%)[a]	0.3 liter/ha	14	3
				Imidacloprid (Provado 1.6F, 17.4%)[a]	0.6 liter/ha	14	3
				Azinphos-methyl (Guthion 50WP, 50%)[c]	1.7 kg/ha	14	2
				and Carbaryl (Sevin XLR Plus, 44.1%)[c]	4.7 liters/ha		1
3	2002	Kaysville, UT	0.4–0.9	Control			
				Imidacloprid (Provado 1.6F, 17.4%)[a]	0.6 liter/ha	14	3
				Azinphos-methyl (Guthion 50WP, 50%)[c]	1.7 kg/ha	14	2
				and Carbaryl (Sevin XLR Plus, 44.1%)[c]	4.7 liters/ha		1
4	2003	Kaysville, UT	0.2	Same as Trial 2			
5	2004	Kaysville, UT	0.3	Control			
				Spinosad Bait (GF-120 NF Naturalyte, 0.02%)[b]	1.5 liters/ha	7	5
				Azinphos-methyl (Guthion 50WP, 50%)[c]	1.7 kg/ha	14	2
				and Carbaryl (Sevin XLR Plus, 44.1%)[c]	4.7 liters/ha		1
6	2005	Kaysville, UT	0.3	Same as Trial 5			
7	2004	Moxee, WA	Single Trees	Control	7.56 liters water/tree	8–10	4
				Spinosad (Entrust,80.0%)[b]	0.34 g/7.56 liters/tree	8–10	4
				Spinosad Bait (GF-120 NF Naturalyte, 0.02%)[b]	88.7 ml/532 ml/tree	8–10	4
				Imidacloprid (Provado, 1.6F, 17.4%)[a]	1.2 ml/7.56 liters/tree	10	3
				Thiacloprid (Calypso, 4F, 40.4%)[a]	1.2 ml/7.56 liters/tree	10	3

[a] Bayer CropScience, Research Triangle Park, NC.
[b] Dow AgroSciences LLC, Indianapolis, IN.
[c] Gowan Company, Yuma, AZ.

randomized complete block design. Each tree was separated from others by one untreated tree. There were seven blocks (replicates). Applications were made within 5 d after the first fly capture. Because of the low fly density, traps were removed afterward to reduce the possibility they would capture too many flies. Spinosad bait was applied using a hand-held sprayer at 532 ml of spray per tree (per label for single trees). The other treatments were applied using a Nifty Pul-Tank (Rear's Mfg Co., Eugene, OR) and a handgun at 100 psi in a volume of 7.56 liters per tree. Applications were made every 8 or 10 d. Two hundred cherries were removed from each tree on 1 July. Mature fruit were laid on emergence trays over 1 mo to collect puparia.

Statistics. In experiments 1 and 2, data were analyzed using one-way analysis of variance (ANOVA), one analysis for each day after exposure. In experiment 3, three-way ANOVA was performed, by using insecticide treatment, topical application or ingestion method of exposure, and sex as fixed factors. In experiment 4, data were analyzed using two-way ANOVA, by using insecticide treatment and insecticide residue age as fixed factors. In experiment 5, one-way ANOVA was conducted, and in experiment 6, randomized complete block or one-way ANOVA was conducted. In all experiments, percentages were subjected to square-root and arcsine-transformation or counts were subjected to square-root (y or y + 1) transformation before analyses to meet normality assumptions. ANOVAs in all experiments except experiment 4 were followed by pairwise comparisons by using Fisher's least significant difference (LSD) test, at α of 0.05. In experiment 4, when insecticide treatment × age residue interactions were significant, simple effects were tested (Schabenberger 1998); when simple effects were significant, LSD tests were conducted. Because of the high numbers of all pairwise comparisons within this experiment, a Bonferroni adjustment for multiplicity was used for 1) different-aged residues within insecticides and 2) comparisons of different insecticides within age residues. For comparison 1, 0.05 was divided by 6, the number of all pairwise comparisons, to give $P = 0.0083$, which was required to declare significance. Similarly, for comparison 2, $\alpha = 0.05$ was divided by 10, the number of all possible comparisons, to give $P = 0.005$, which was required to declare significance. SAS version 8 for Windows (SAS Institute 2001) was used for all analyses. Means ± SE are reported.

Results

Experiment 1: Effects of Insecticides and Bait on Adult Mortality. In test 1A using 100-μl solutions, mortality among all treatments was higher than in the control, and rankings of effectiveness were similar among days (Table 2). Spinosad caused the highest mortality, although statistically it was not different from the spinosad bait. Imidacloprid did not differ

Table 2. Effects of insecticides and bait on mean cumulative percentage of mortality ± SE of adult *R. indifferens* at 1–4 d after exposure

Treatment	Test 1A: 100-μl solution			
	1 d	2 d	3 d	4 d
Control	2.5 ± 2.5c	3.8 ± 3.8c	8.8 ± 2.4c	16.3 ± 8.3c
Spinosad	77.0 ± 9.0a	94.8 ± 2.2a	100.0 ± 0.0a	100.0 ± 0.0a
Spinosad bait	57.3 ± 9.1ab	79.3 ± 10.9ab	89.7 ± 8.0ab	96.3 ± 1.9ab
Imidacloprid	40.5 ± 8.9b	58.5 ± 17.4ab	63.3 ± 16.3b	68.5 ± 17.6b
Thiacloprid	45.0 ± 10.4b	50.0 ± 15.3b	60.0 ± 20.8b	63.3 ± 20.3b
One-way ANOVA				
(df = 4, 13) *F*	16.17	9.66	8.89	7.76
P	<0.0001	0.0007	0.0011	0.0020
	Test 1B: 500-μl solution			
Treatment	1 d	2 d	3 d	4 d
Control	0.0 ± 0.0c	0.0 ± 0.0c	2.0 ± 1.2c	5.0 ± 2.2c
Spinosad	75.0 ± 5.7a	88.0 ± 4.9a	99.0 ± 1.0a	100.0 ± 0.0a
Spinosad bait	70.2 ± 8.8a	86.6 ± 7.5a	93.8 ± 4.1a	97.0 ± 3.0a
Imidacloprid	50.0 ± 9.1ab	65.0 ± 6.9b	87.0 ± 8.9a	89.0 ± 7.1a
Thiacloprid	38.0 ± 14.9b	43.0 ± 15.1b	60.0 ± 13.5b	65.0 ± 13.3b
One-way ANOVA				
(df = 4, 20) *F*	16.30	21.25	3.56	26.20
P	<0.0001	<0.0001	<0.0001	<0.0001

Test 1A, three or four replicates; test 1B, five replicates; both 20 flies per replicate. Flies were exposed to insecticides throughout 4 d.
Means followed by the same letter within columns are not significantly different (LSD test; $P > 0.05$).

from spinosad bait and thiacloprid, but it was less effective than spinosad at 1, 3, and 4 d, and thiacloprid was less effective than spinosad at 1, 2, 3, and 4 d (Table 2). Exposure to 5 times greater volume in test 1B resulted in higher mortality in the imidacloprid than thiacloprid treatment at 3 and 4 d. Mortality caused by spinosad or spinosad bait and imidacloprid was not different except at 2 d after exposure (Table 2).

Experiment 2: Effects of Insecticide- and Bait-Treated Cherries on Adult Mortality and Oviposition. In test 2A, mortality of flies exposed to cherries dipped in spinosad and spinosad bait was greater than that of flies exposed to cherries dipped in imidacloprid and thiacloprid at 1–4 d (Table 3). Mortality in the imidacloprid treatment was greater than in the thiacloprid treatment at 2 d, but the two treatments were not different at 1, 3, and 4 d (Table 3). No eggs were laid in cherries treated with spinosad and spinosad bait, whereas eggs were laid in control, imidacloprid-, and thiacloprid-treated cherries (Table 3). In test 2B, mortality of flies exposed to cherries dipped in the four materials followed the pattern in test 2A, except by 6 d, imidacloprid was as effective as spinosad and spinosad bait (Table 3). No eggs were laid in cherries treated with spinosad bait, but eggs were laid in all other

Table 3. Effects of insecticide- and bait-treated cherries on mean cumulative percent mortality ± SE of adult *R. indifferens* and oviposition (mean eggs per female) ± SE at 1–4 or 1, 2 and 6 d after exposure to treated cherries

Treatment	Test 2A				
	1 d	2 d	3 d	4 d	Eggs/♀/3 fruit
Control	0.0 ± 0.0c	0.0 ± 0.0d	11.7 ± 7.4c	23.3 ± 10.5c	23.0 ± 3.8a
Spinosad	98.3 ± 1.1a	100.0±0.0a	100.0 ± 0.0a	100.0 ± 0.0a	0.0 ± 0.0b
Spinosad bait	94.2 ± 2.4a	99.2 ± 0.8a	100.0 ± 0.0a	100.0 ± 0.0a	0.0 ± 0.0b
Imidacloprid	55.2 ± 8.2b	66.2 ± 8.2b	78.0 ± 6.6b	83.7 ± 5.8b	3.4 ± 1.6b
Thiacloprid	46.3 ± 7.8b	49.7 ± 6.5c	64.8 ± 5.9b	65.7 ± 5.3b	15.2 ± 4.0a
One-way ANOVA					
(df = 4, 25) *F*	85.49	136.09	47.54	30.16	23.09
P	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
	Test 2B				
Treatment	1 d	2 d	6 d	Eggs/♀/3 fruit[a]	No. larvae[a]
Control	0.0 ± 0.0c	3.3 ± 2.1d	14.0 ± 2.4c	9.7 ± 4.8	6.7 ± 1.8a
Spinosad	81.7 ± 5.4a	96.7 ± 2.1a	100.0 ± 0.0a	1.3 ± 0.3	0.7 ± 0.7b
Spinosad bait	93.3 ± 2.1a	100.0 ± 0.0a	100.0 ± 0.0a	0.0 ± 0.0	0.0 ± 0.0b
Imidacloprid	16.7 ± 3.3b	40.0 ± 3.7b	98.3 ± 1.7a	1.7 ± 1.5	0.0 ± 0.0b
Thiacloprid	10.0 ± 3.7b	21.7 ± 4.8c	45.0 ± 10.9b	2.9 ± 1.2	0.0 ± 0.0b
One-way ANOVA					
(df = 4, 25) *F*	77.09	144.24	77.89	3.24	17.96
P	<0.0001	<0.0001	<0.0001	0.0598	<0.0001

Test 2A: six replicates, 20 flies per replicate; test 2B: six replicates, 10 flies per replicate.
Means followed by the same letter within columns are not significantly different (LSD test; $P > 0.05$).
[a] Each variable evaluated from three of six replicates.

Table 4. Effects of topical application and ingestion of insecticides on mean days survived ± SE posttreatment by single adult *R. indifferens*

Treatment	Topical application of water or insecticide[a]				Ingestion of water or insecticide[b]				Overall means insecticides
	n	Males	*n*	Females	*n*	Males	*n*	Females	
Water	17	19.1 ± 3.0	25	15.5 ± 2.4	17	16.1 ± 3.0	18	17.3 ± 3.1	16.8 ± 1.4a
Spinosad	18	4.9 ± 2.2	20	2.8 ± 1.4	15	1.0 ± 0.0	17	1.2 ± 0.2	2.6 ± 0.7c
Imidacloprid	15	7.5 ± 2.4	18	10.2 ± 2.4	17	4.3 ± 2.1	18	7.0 ± 2.4	7.3 ± 1.2b
Thiacloprid	16	13.3 ± 3.1	18	12.3 ± 2.9	17	6.1 ± 2.3	19	4.3 ± 2.1	8.8 ± 1.4b
Three-way ANOVA	Effect	Num. df	Den. df	*F*	*P*				
	Insecticide treatment	3	269	26.03	<0.0001				
	Exposure method	1	269	8.74	0.0034				
	Sex	1	269	0.04	0.8499				
	Treatment × method	3	269	1.54	0.2035				
	Treatment × sex	3	269	0.63	0.5931				
	Method × sex	1	269	0.45	0.5042				
	Treatment × method × sex	3	269	0.28	0.8388				

Water and all treatments contained 20% sucrose (wt:wt); followed for maximum of 30 d; flies 3–7 d old at treatment.
Overall means (of insecticides) followed by the same letter are not significantly different (LSD test; $P > 0.05$).
[a] Two-microliter drop applied on top of thorax.
[b] Two-microliter drop exposed to flies; all flies fed, but drop not always entirely consumed.

treatments, with most in the control, although differences were not significant (Table 3). However, numbers of larvae were greater from the control than from all treatments (Table 3).

Experiment 3: Effects of Topical Application and Ingestion of Insecticides on Adult Mortality. Insecticides differed in their ability to kill adults, as evidenced by differential longevity, through topical application or ingestion (Table 4). Three-way ANOVA indicated ingestion of insecticides (7.3 ± 0.8 d) significantly reduced the longevity of flies compared with topical application (10.8 ± 1.0 d) and that spinosad reduced longevity greater than imidacloprid and thiacloprid, with no differential sex effects (Table 4).

Experiment 4. Effects of Aging Insecticide and Bait Residues on Adult Mortality. There were significant effects of aging insecticides and baits and of insecticide and bait residues on adult mortality (Table 5). Two-way ANOVA indicated there were significant residue age × insecticide treatment interactions, because not all treatments decreased in effectiveness or in the same way as they aged: there was no age effect for the control, a significant age effect for spinosad, no age effect for spinosad bait, a significant age effect for imidacloprid, and no age effect for thiacloprid at 1, 3, and 7 days after exposure (DAE) (Table 5). Spinosad aged 14 d was less effective than spinosad aged 3 or 7 d. Spinosad bait aged 0–14 d was similarly effective. Imidacloprid aged 14 d was less effective than imidacloprid aged 0 or 3 d. Thiacloprid aged 0–14 d was equally ineffective (Table 5).

Compared with the control, one or more insecticides or bait were effective within every residue age at 1, 3, and 7 DAE (Table 5). At 1 DAE, mortality within 0-d-old residues was similar with spinosad and spinosad bait and greater than with thiacloprid. At 1 DAE, mortality within 3-, 7-, and 14-d-old residues was significant only with spinosad bait. Spinosad, imidacloprid, and thiacloprid did not differ from the control. Similar patterns were seen at 3 and 7 DAE. The main difference was that spinosad did not differ from spinosad bait except when both were aged 14 d. Spinosad bait was the only material that caused 100% mortality at 7 DAE when aged 0, 3, or 14 d (Table 5).

Experiment 5: Effects of Spraying Cherries with Insecticides and Bait on Larval Mortality and Emergence from Fruit. There were few dead larvae inside the cherries at 8 d after treatment, and no differences in dead larval numbers among the control and any treatments were detected (Table 6). The dead larvae were 2.1–6.8 mm in length, but there were too few deaths to determine whether growth had been affected by insecticides. However, the numbers of live larvae were similar in the control, spinosad, and spinosad bait treatments, but they were significantly lower in the imidacloprid and thiacloprid treatments (Table 6). Larvae were similar in length, suggesting no effect on growth. Larval emergence from fruit was lower in all four treatments than in the control at days 1–15. At days 16–30, emergence was lowest from the imidacloprid and thiacloprid treatments, although thiacloprid was not different from the spinosad bait. Spinosad was not different from the control. Over the 30 d, all treatments had significantly lower emergence than the control, with imidacloprid having the lowest numerically, even though it was not different from the spinosad bait (Table 6). There was no effect of any material on mortality of pupae at 30–37 d posttreatment (Table 6).

Experiment 6: Effects of Insecticide and Bait Sprays on Adult and Larval Control in the Field. Numbers of adults caught on traps in trials 1–6 in Utah are shown in Table 7. The numbers of adults in insecticide and bait treatments were lower than in the controls in five of six trials. Spinosad (reapplied every 7 d), spinosad bait, and imidacloprid treatments had similar adult numbers as azinphos-methyl, but the thiacloprid treat-

Table 5. Effects of field-aged insecticide and bait residues on leaves (6–20 June 2005) on mean cumulative percentage mortality ± SE of adult *R. indifferens* in the laboratory at 1, 3, and 7 DAE

	Age of residues on cherry leaves at initial exposure to flies			
	0 d (fresh)	3 d	7 d	14 d
1 DAE				
Control	3.3 ± 2.0 (d)	1.7 ± 1.7 (b)	3.5 ± 2.1 (b)	1.7 ± 1.7 (b)
Spinosad	70.0 ± 8.2a (ab)	26.0 ± 13.8b (b)	14.6 ± 6.5bc (b)	0.0 ± 0.0c (b)
Spinosad bait	78.3 ± 10.1 (a)	72.4 ± 9.5 (a)	70.1 ± 12.8 (a)	79.4 ± 6.9 (a)
Imidacloprid	34.2 ± 6.6a (bc)	17.0 ± 4.5ab (b)	23.0 ± 9.4ab (b)	5.0 ± 3.3b (b)
Thiacloprid	8.3 ± 4.6 (cd)	3.5 ± 2.1 (b)	8.8 ± 5.6 (b)	13.3 ± 5.0 (b)
3 DAE	0 d (fresh)	3 d	7d	14 d
Control	10.1 ± 1.6 (c)	3.3 ± 2.0 (c)	8.6 ± 2.6 (c)	3.3 ± 2.0 (b)
Spinosad	95.0 ± 3.3a(a)	76.7 ± 19.4a (a)	65.0 ± 11.0a (ab)	8.3 ± 3.7b (b)
Spinosad bait	98.3 ± 1.7 (a)	100.0 ± 0.0 (a)	91.1 ± 7.1 (a)	96.9 ± 3.1 (a)
Imidacloprid	63.0 ± 8.4 (b)	42.3 ± 10.2ab (b)	32.3 ± 11.4ab (bc)	11.7 ± 6.2b (b)
Thiacloprid	16.7 ± 5.3 (c)	19.3 ± 3.2 (bc)	8.8 ± 5.6 (c)	26.7 ± 8.1 (b)
7 DAE	0 d (fresh)	3 d	7d	14 d
Control	23.0 ± 3.0 (b)	17.6 ± 2.7 (c)	20.4 ± 9.6 (b)	15.0 ± 4.9 (b)
Spinosad	98.3 ± 1.7a (a)	88.3 ± 11.7a (a)	98.3 ± 1.7a (a)	17.2 ± 7.5b (b)
Spinosad bait	100.0 ± 0.0 (a)	100.0 ± 0.0 (a)	98.2 ± 1.8 (a)	100.0 ± 0.0 (a)
Imidacloprid	91.7 ± 4.6 a (a)	57.4 ± 11.1b (b)	41.0 ± 9.7bc (b)	23.3 ± 6.7c (b)
Thiacloprid	31.7 ± 7.2 (b)	35.1 ± 7.6 (bc)	23.8 ± 4.2 (b)	35.0 ± 10.7 (b)

		Factor	*F*	df	*P*
1 DAE					
	Two-way ANOVA	Age residues	6.39	3, 80	0.0006
		Insecticide/bait treatment	50.90	4, 80	<0.0001
		Age residues × insecticide/bait	3.38	12, 80	0.0005
3 DAE					
	Two-way ANOVA	Age residues	10.44	3, 80	<0.0001
		Insecticide/bait treatment	76.39	4, 80	<0.0001
		Age residues × insecticide/bait	4.32	12, 80	<0.0001
7 DAE					
	Two-way ANOVA	Age residues	20.47	3, 80	0.0001
		Insecticide/bait treatment	107.22	4, 80	<0.0001
		Age residues × insecticide/bait	9.22	12, 80	<0.0001

Test of insecticide/bait treatment × age residues simple effects

			1 DAE		3 DAE		7 DAE	
Age effects	Num. df	Den. df	*F*	*P*	*F*	*P*	*F*	*P*
Control	3	80	0.10	0.9609	0.80	0.4990	0.33	0.8071
Spinosad	3	80	15.77	<0.0001	19.66	<0.0001	39.14	<0.0001
Spinosad bait	3	80	0.20	0.8968	0.45	0.7157	0.12	0.9481
Imidacloprid	3	80	3.25	0.0260	5.56	0.0016	17.40	<0.0001
Thiacloprid	3	80	0.60	0.6168	1.25	0.2960	0.36	0.7820
Insecticide/bait effects								
0 d (fresh)	4	80	17.89	<0.0001	23.27	<0.0001	34.34	<0.0001
3 d	4	80	13.34	<0.0001	24.49	<0.0001	30.80	<0.0001
7 d	4	80	11.53	<0.0001	18.54	<0.0001	36.97	<0.0001
14 d	4	80	18.29	<0.0001	23.05	<0.0001	32.76	<0.0001

Five replicates of 12 flies each (eight males, four females).

Means followed by the same letter within rows without parentheses or within columns inside parentheses are not significantly different (ANOVA, LSD test with Bonferroni adjustment; different age residues within insecticides, $P > 0.0083$; different insecticides within age residues, $P > 0.005$).

ment had significantly higher numbers than imidacloprid and azinphos-methyl treatments in one trial with high fly densities (trial 4) (Table 7).

Numbers of larvae per 100 immature and/or 100 mature fruit in trials 1–7 are shown in Table 8. Numbers of larvae in spinosad, spinosad bait, imidacloprid, or azinphos-methyl and carbaryl treatments were similar and were lower than in controls in trials 1, 3, and 5–7 in immature or mature fruit. The number in mature fruit in the imidacloprid treatment was higher than in the azinphos-methyl and carbaryl treatment in trial 3, and numbers in immature or mature fruit in the thiacloprid treatment were higher than in imidacloprid and azinphos-methyl and carbaryl treatments in trials 2 and 4 (Table 8).

Discussion

The results of experiment 1 indicate that at the high label rate, spinosad is highly toxic and more effective than imidacloprid or thiacloprid in causing mortality in adult *R. indifferens.* Surprisingly, spinosad and spinosad bait caused similar mortality, suggesting the bait in the laboratory did not enhance mortality by causing flies to feed on it quickly after exposure. Neither imidacloprid nor thiacloprid was especially toxic to adults before 1 d (<50% mortality), so the higher mortality at 4 d was probably caused by a delayed effect or repeated contact with the toxins. The effectiveness of spinosad, imidacloprid, and thiacloprid against *R. indifferens* seems similar to that against *R. pomonella* and

Table 6. Effects of spraying cherries with insecticides and bait on larval mortality ± SE and numbers of puparia ± SE of *R. indifferens* from cherries collected in Kennewick, WA, 2005

Treatment	Dead larvae/10 fruit[a]		Live larvae/10 fruit[a]	
	No.	Length (mm)[b]	No.	Length (mm)
Control	0.2 ± 0.2	6.8	12.6 ± 1.5a	4.9 ± 0.4
Spinosad	1.0 ± 1.0	4.5	12.0 ± 1.8a	4.2 ± 0.3
Spinosad bait	0.8 ± 0.5	4.6 ± 0.6	11.4 ± 2.9a	4.2 ± 0.4
Imidacloprid	1.2 ± 0.4	3.5 ± 1.7	6.4 ± 0.5b	3.8 ± 0.2
Thiacloprid	0.8 ± 0.4	2.1 ± 0.4	6.4 ± 0.7b	4.9 ± 0.3
One-way ANOVA				
(df = 4, 20) *F*	0.56		3.86	2.28
P	0.6959		0.0175	0.0962
	No. puparia/100 fruit			
Treatment	Days 1–15	Days 16–30	30-d Total	% pupae dead[c]
Control	130.8 ± 12.5a	44.4 ± 5.8a	175.2 ± 10.8a	36.9 ± 3.2
Spinosad	70.4 ± 10.3b	33.3 ± 6.9ab	103.4 ± 11.6b	39.6 ± 6.4
Spinosad bait	66.0 ± 5.5b	18.6 ± 3.9bc	84.6 ± 7.9bc	40.4 ± 6.1
Imidacloprid	58.2 ± 13.6b	6.0 ± 3.6d	64.2 ± 11.2c	42.3 ± 11.1
Thiacloprid	89.0 ± 15.7b	12.0 ± 7.4cd	101.0 ± 16.7b	39.4 ± 6.6
One-way ANOVA				
(df = 4, 20) *F*	5.46	7.87	10.69	0.03
P	0.0039	0.0006	<0.0001	0.9976

Five replicates of the control and each treatment.
Means followed by the same letter within days posttreatment are not significantly different (LSD test; $P > 0.05$).
[a] At 8 d posttreatment; one application only.
[b] Too few to analyze statistically.
[c] At 30–37 d posttreatment after larvae emerged.

R. mendax in the laboratory (Reissig 2003, Barry and Polavarapu 2005, Barry et al. 2005).

The results of experiment 2 showed that spinosad bait on cherries prevented all oviposition, whereas spinosad, imidacloprid, and thiacloprid did not. Spinosad in the bait thus killed the females before they had a chance to oviposit. However, in another study, spinosad bait did not kill flies quickly enough to prevent oviposition (Yee and Chapman 2005), so its ability to kill quickly seems inconsistent. In *R. mendax* and *R. pomonella*, spinosad, imidacloprid, and thiacloprid also failed to prevent oviposition or puncturing of fruit (Liburd et al. 2003, Reissig 2003). Even azinphos-methyl, although a strong oviposition deterrent, did not prevent *R. pomonella* from puncturing apples (Reissig et al. 1983). In our study, eggs or larvae inside the spinosad-, imidacloprid-, and thiacloprid-treated cherries were probably killed by the insecticides, because few or no larvae emerged from treated cherries.

The results of experiment 3 show that insecticides were more effective when ingested than when topically applied. This suggests there was penetration of the insecticides through both the cuticle and the digestive system. For spinosad, this seems contrary to the idea that this insecticide has no or little contact toxicity against flies (Moreno and Mangan 2003), although it is possible grooming of small amounts of runoff from the thorax may have resulted in oral contact and ingestion. Likewise, imidacloprid and thiacloprid were more effective when ingested than topically applied, suggesting they had no or little contact activity at the dose tested. Differences in effects may

Table 7. Effects of insecticide and bait sprays on numbers of *R. indifferens* adults caught per trap ± SE from mid-May to early or mid-August in efficacy trials in cherry trees, Utah, 2000–2005

Treatment	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5	Trial 6
Control	17.7 ± 0.9a	248.8 ± 14.3	321.5 ± 37.9a	1038.0 ± 95.5a	1210.8 ± 123.8a	464.3 ± 43.9a
Spinosad (Success) 14 d	9.0 ± 0.3ab					
Spinosad (Success) 7 d	6.7 ± 0.2b					
Spinosad bait					186.8 ± 25.8b	48.3 ± 4.7b
Imidacloprid	4.7 ± 0.5b	272.0 ± 14.6	193.7 ± 22.3b	362.3 ± 26.4c		
Thiacloprid	6.3 ± 0.8b	229.3 ± 19.0		611.3 ± 39.5b		
Azinphos-methyl and carbaryl	4.3 ± 0.5b	195.5 ± 19.4	64.2 ± 11.7c	208.3 ± 30.2c	248.5 ± 62.8b	68.8 ± 12.9b
ANOVA[a]						
F	6.62	0.52	5.94	42.44	54.33	92.09
df	5, 15	3, 9	2, 15	3, 12	2, 15	2, 9
P	0.0057	0.6761	0.0032	<0.0001	<0.0001	<0.0001

Means followed by the same letter within columns are not significantly different (LSD test; $P > 0.05$). No trapping conducted for adult flies in trial 7.
[a] Trials 1 and 2, randomized complete block ANOVA; trials 3–6, one-way ANOVA.

Table 8. Effects of insecticide and bait sprays on numbers of *R. indifferens* larvae per 100 cherries ± SE in immature fruits (collected mid- to late June) and mature fruits (collected late June to mid-July) in efficacy trials in Utah and Washington, 2000–2005

Treatment	Trial 1		Trial 2		Trial 3	
	Immature	Mature	Immature	Mature	Immature	Mature
Control	0	0.7±0.3a	1.2±0.4a	2.8±0.9a	36.5±9.4a	4.1±15.2a
Spinosad (Success) 14 d	0	0b				
Spinosad (Success) 7 d	0	0b				
Imidacloprid	0	0b	0b	0c	0b	0.01 ± 0.01b
Thiacloprid	0	0b	0b	0.1 ± 0.03b		
Azinphos-methyl and carbaryl	0	0b	0b	0c	0b	0c
ANOVA[a]						
F		4.78	25.00	13.67	93.29	117.06
df	5, 15	5, 40	3, 9	3, 41	2, 15	2, 51
P		0.0013	<0.0001	<0.0001	<0.0001	<0.0001

Treatment	Trial 4		Trial 5		Trial 6		Trial 7
	Immature	Mature	Immature	Mature	Immature	Mature	Mature[b]
Control	35.3 ± 1.0a	20.6 ± 3.0a	49.7 ± 8.1a	44.7 ± 5.2a	9.4 ± 2.2a	9.3 ± 2.4a	2.45 ± 0.9a
Spinosad (Entrust)							0.2 ± 0.2b
Spinosad bait			0b	0.3 ± 0.1b	0.6 ± 0.4b	0.1 ± 0.1b	0b
Imidacloprid	0c	0c					0.1 ± 0.1b
Thiacloprid	3.0 ± 2.0b	2.7 ± 0.6b					0.2 ± 0.2b
Azinphos-methyl and carbaryl	0c	0c	0b	1.1 ± 0.8b	0b	1.3 ± 0.8b	
ANOVA[a]							
F	85.87	82.22	62.55	212.39	21.27	16.47	6.35
df	3, 12	3, 60	2, 15	2, 51	2, 21	2, 21	4, 24
P	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0002	0.0012

Means followed by the same letter within columns are not significantly different (LSD test; $P > 0.05$).

[a] Trials 1, 2, and 7, randomized complete block ANOVA; trials 3–6, one-way ANOVA.

[b] No immature fruits collected.

exist between tarsal contact with insecticides through walking and topical application of insecticides. Comparisons among insecticides topically applied indicate that spinosad was the most toxic material, consistent with results from previous experiments.

The results of experiment 4 indicate that residual toxicities of materials varied significantly and that spinosad and spinosad bait were overall the most toxic materials. At 1 DAE, spinosad aged 3 d lost effectiveness, as mortality caused by it was no higher than in the control. Furthermore, mortality caused by spinosad went from 70.0 to 26.0% after just 3 d. These results suggest spinosad needs protectants to prevent breakdown from UV rays. In contrast, spinosad bait was effective even after aging for 14 d, suggesting no degradation of its toxic properties, perhaps due to protection by binding agents in the bait, similar to or the same as those in its precursor Solbait (Moreno and Mangan 2003). However, against *R. completa,* spinosad bait aged for only 3 d in California lost most of its toxicity (Van Steenwyk et al. 2003). Possibly the lower bait concentration used in that study compared with ours (20 versus 40%) or possible humidity differences caused the inconsistency. At 1 DAE, imidacloprid aged 3 d lost effectiveness, because mortality caused by it was similar to that in the control. When aged 14 d, its effects were even less, suggesting that it had degraded or that it was absorbed into the leaf, making it unavailable to flies for contacting or ingesting. Consistent with previous experiments, thiacloprid was the least effective of the materials. For all materials, increased encounters with the treated leaves over the 7 d of the experiment and normal fly aging probably accounted for the higher mortality over time. Results clearly indicate application frequencies of the different materials need to differ to produce similar levels of mortality. The greater overall mortality in spinosad and spinosad bait treatments than in imidacloprid and thiacloprid treatments showed toxicities of materials differed, consistent with results from previous experiments.

In experiment 5, spraying cherries with spinosad, spinosad bait, imidacloprid, and thiacloprid did not result in higher numbers of dead large larvae, but imidacloprid- and thiacloprid-treated cherries had significantly fewer live larvae than other cherries, and all materials reduced larval emergence. There were few exit holes when fruit were collected, so it seemed materials moved through the tissues of the fruit. Spinosad is known to be absorbed by plant tissue (Thompson et al. 1999, Dow AgroSciences 2004). Based on numbers of live larvae inside cherries at day 8 posttreatment, imidacloprid and thiacloprid may have moved through the fruit more quickly than spinosad and killed eggs or larvae. Whereas imidacloprid and thiacloprid were expected to reduce larval emergence the most, spinosad and spinosad bait overall performed as well as imidacloprid or thiacloprid. Possibly dissections conducted at 8 d detected mortality mostly of first instars, and the toxicity of imidacloprid and thiacloprid weakened afterward. Toxicity of spinosad may have weakened more, based on similar larval emergence from all treated cherries at days 1–15 but greater larval emergence from spinosad-treated cherries at days 16–30. Multiple sprays on unripe fruit, which have the highest proportion of eggs (Yee 2005), may make all materials more effective than the single spray performed on the ripe fruit. Effectiveness of materials sprayed on fruit on trees and on detached fruit may differ and needs to be compared. Results suggest some materials, especially imidacloprid, can possibly replace the organophosphate Dimethoate as a postharvest spray. It seemed survival of the pupae that were exposed to insecticides inside fruit as larvae were not affected over the short term, but it is unknown whether all larvae contacted the insecticides.

The results of experiment 6 showed all materials significantly suppressed adult and larval *R. indifferens* populations in the field and that control levels followed expectations based on laboratory studies. In addition, spinosad, spinosad bait, and imidacloprid were usually statistically comparable with azinphos-methyl and carbaryl in effectiveness against larvae. Because of their toxicity to adults, spinosad and spinosad bait probably killed most if not all the flies that contacted or fed on them before oviposition occurred, explaining the low larval infestations. Because of its apparent lower toxicity to adults, imidacloprid was expected to be less effective than spinosad or spinosad bait. However, all were equally effective in controlling larvae, and imidacloprid was as effective as azinphos-methyl in six of seven comparisons, perhaps because imidacloprid killed adults as well as eggs or larvae (evidence in experiments 2 and 5). Thiacloprid had low toxicity against adults in the laboratory, explaining its relatively poor performance in controlling larvae in the field compared with imidacloprid and with azinphos-methyl and carbaryl (less effective in three of five comparisons in both cases). Thiacloprid probably suppressed larval numbers mostly because of its toxic effects on eggs and larvae. The failure of spinosad and spinosad bait and sometimes even azinphos-methyl and carbaryl to eliminate infestations suggests sprays of these materials at the volumes tested cannot protect fruit against dispersing, mature flies, which are a problem when population densities in surrounding trees are high. Against *R. mendax*, spinosad, spinosad bait, and imidacloprid did not eliminate larval infestations in blueberries, but they did cause 94–99.7% reductions compared with the control (Barry et al. 2005).

Our overall results show that spinosad and chloronicotinyl insecticides differed significantly in their effectiveness against adults and larvae of *R. indifferens.* Specifically, spinosad and spinosad bait were more effective against adults but less effective against larvae than the chloronicotinyl insecticides. Spinosad, spinosad bait, and imidacloprid seem to be acceptable substitutes for organophosphate and carbamate insecticides for controlling *R. indifferens,* with spinosad bait apparently the most effective due to its high and long residual toxicity. Given the zero tolerance for larval infestations and our combined laboratory and field results, thiacloprid does not seem to be an acceptable substitute at the recommended application rate. Future research should focus on elucidating the modes of action of these and other insecticides on the different fly life stages, determining the effects of insecticides on immature stages in fruit of different maturity, and on testing insecticide chemistries or bait mixes that target dispersing flies and that are capable of preventing oviposition. Combining laboratory and field research may result in even safer and more effective chemistries and better application methods for controlling *R. indifferens* and other tephritid species.

Acknowledgments

We thank Julie Schlaman, Janine Jewett, and Pete Chapman (USDA–ARS) and Sadie Enright, Bruno Galli, Jessica Rowley, and Mylie Thompson (Utah State University) for laboratory and field assistance and Mark Hertlein, Barat Bisabri, Harvey Yoshida (Dow AgroSciences) and Dean Christie, Ron Brenchley, and Dennis Scott (Bayer CropScience) for supplying insecticides. We also thank Bruce Mackey and David Horton (USDA–ARS) for advice on statistics and David Horton, Bob Van Steenwyk (University of California, Berkeley), and Mike Bush (Washington State University) for reviewing the manuscript. This work was funded in part by the Washington Tree Fruit Research Commission, the Utah State Horticultural Association Research Committee, and the Utah Agricultural Experiment Station.

References Cited

Anonymous. 1968. Permanent order no. 1099 prohibiting the sale and/or movement of infested cherries. Washington Administrative Code 16-463-001, 16-463-010. Effective 30 September 1968. (http://www.leg.wa.gov/).

Barry, J. D., and S. Polavarapu. 2005. Feeding and survivorship of blueberry maggot flies (Diptera: Tephritidae) on protein baits incorporated with insecticides. Fla. Entomol. 88: 268–277.

Barry, J. D., W. J. Sciarappa, L.A.F. Teixeira, and S. Polavarapu. 2005. Comparative effectiveness of different insecticides for organic management of blueberry maggot (Diptera: Tephritidae). J. Econ. Entomol. 98: 1236–1241.

Dow AgroSciences. 2004. Spinosad technical bulletin. Dow AgroSciences, Indianapolis, IN.

Eide, P. M., F. J. Lynd, and H. S. Telford. 1949. The cherry fruitfly in eastern Washington. Washington State Agric. Expt. Stn. Circ. 72: 1–8 (F).

Elbert, A., B. Becker, J. Hartwig, and C. Erdelen. 1991. Imidacloprid – a new systemic insecticide. Pflanzenschutz-Nachrichten Bayer 44: 113–136.

Elbert, A., A. Buchholz, U. Ebbinghaus-Kintscher, C. Erdelen, R. Nauen, and H.-J. Schnorbach. 2001. The biological profile of thiacloprid – a new chloronicotinyl insecticide. Pflanzenschutz-Nachrichten Bayer 54: 185–208.

Food Quality and Protection Act. 1996. U.S. Congressional Record, vol. 142: 1489–1538.

Frick, K. E. 1957. Comparative toxicity tests as an aid in selecting insecticides for control of the cherry fruit fly. J. Econ. Entomol. 50: 256–259.

Frick, K. E., and H. G. Simkover. 1953. Insecticides for killing cherry fruit fly maggots within the fruit. J. Econ. Entomol. 46: 361–362.

Frick, K. E., H. G. Simkover, and H. S. Telford. 1954. Bionomics of the cherry fruit flies in eastern Washington. Wash. Agric. Exp. Stn. Tech. Bull. 13.

Hu, X. P., A. Kaknes, and R. J. Prokopy. 1998. Can apple maggot fly control benefit from spray of Provado aimed at killing leafminers and leafhoppers? Fruit Notes 63: 4–6.

Hu, X. P., R. J. Prokopy, and J. M. Clark. 2000. Toxicity and residual effectiveness of insecticides on insecticide-treated spheres for controlling females of *Rhagoletis pomonella* (Diptera: Tephritidae). J. Econ. Entomol. 93: 403–411.

Johansen, C. A., W. E. Westlake, L. I. Butler, and R. E. Bry. 1954. Residual action and toxicity of methoxychlor and parathion to the cherry fruit fly. J. Econ. Entomol. 47: 746–749.

Liburd, O. E., E. M. Finn, K. L. Pettit, and J. C. Wise. 2003. Response of blueberry maggot fly (Diptera: Tephritidae) to imidacloprid-treated spheres and selected insecticides. Can. Entomol. 135: 427–438.

Moreno, D. S., and R. L. Mangan. 2003. Bait matrix for novel toxicants for use in control of fruit flies (Diptera: Tephritidae), pp. 333–362. *In* C. Schwalbe [ed.], Invasive arthropods in agriculture. Science Publishers, Inc., Enfield, NH.

Raine, J., and H. Andison. 1958. Life histories and control of cherry fruit flies on Vancouver Island, British Columbia. J. Econ. Entomol. 51: 592–595.

Reissig, W. H. 2003. Field and laboratory tests of new insecticides against the apple maggot, *Rhagoletis pomonella* (Walsh) (Diptera: Tephritidae). J. Econ. Entomol. 96: 1463–1472.

Reissig, W. H., B. H. Stanley, M. E. Valla, R. C. Seem, and J. B. Bourke. 1983. Effects of surface residues of azinphosmethyl on apple maggot behavior, oviposition, and mortality. Environ. Entomol. 12: 815–822.

SAS Institute. 2001. SAS/STAT user's guide, version 8. SAS Institute, Cary, NC.

Schabenberger, O. 1998. Slicing interactions in SAS. SAS Institute, Cary, NC. (http:// www. stat.vt.edu/~oliver/SASSlice.html).

Thompson, G. D., S. H. Hutchins, and T. C. Sparks. 1999. Development of spinosad and attributes of a new class of insect control products. University of Minnesota National IPM Network. (http://ipmworld.umn.edu/chapters/hutchins2.htm).

Van Steenwyk, R. A., and W. W. Coates. 1998. Walnut husk fly control with low-toxicity insecticides, 1997. Arthropod Manage. Tests 23: 72.

Van Steenwyk, R. A., S. K. Zollbrod, and R. M. Nomoto. 2003. Walnut husk fly control with reduced risk insecticides. *In* Proceedings of the 77th Annual Western Orchard Pest & Disease Management Conference, 15–17 January 2003, Portland, OR. Washington State University, Pullman, WA.

Yee, W. L. 2005. Seasonal distributions of eggs and larvae of *Rhagoletis indifferens* Curran (Diptera: Tephritidae) in cherries. J. Entomol. Sci. 40: 158–166.

Yee, W. L., and P. S. Chapman. 2005. Effects of GF-120 Fruit Fly bait concentrations on attraction, feeding, mortality, and control of *Rhagoletis indifferens* (Diptera: Tephritidae). J. Econ. Entomol. 98: 1654–1663.

Zwick, R. W., S. C. Jones, F. W. Peifer, R. W. Every, R. L. Smith, and J. R. Thienes. 1970. Malathion ULV aerial applications for cherry fruit fly control. J. Econ. Entomol. 63: 1693–1695.

Zwick, R. W., G. J. Fields, and U. Kiigemagi. 1975. Dimethoate for control of western cherry fruit fly on sweet cherry in Oregon. J. Econ. Entomol. 68: 383–385.

Received 6 January 2006; accepted 24 June 2006.